speech, instability of mood, and excessive words all imply a disinhibition of impulses and affective processes. The term dysmentia does not seem quite as applicable as dyscontrol or disinhibition. The probability that these symptoms are reflecting a illness-related syndrome rather than a neuroleptic-related syndrome has not been ruled out. This might be difficult to do as chronic schizophrenic patients who have not been exposed to long-term neuroleptic treatment are virtually impossible to find. On the other hand, there are groups of patients with long-term neuroleptic exposure in whom the natural course of the psychiatric illness does not suggest a dysmentia/dyscontrol syndrome developing. An example would be the case of bipolar disorder. We have studied TD in a cohort of 131 bipolar patients (Mukherjee et al., submitted for publication). The occurrence of TD was limited to the patients with long-term neuroleptic exposure history. However, none of these patients showed the type of behavioral picture proposed as being characteristic of tardive dysmentia during periods of remission from affective episodes. This does not support tardive dysmentia (dyscontrol?) as being a neuroleptic-induced neurotoxic phenomenon. However, it does not rule it out either. It may quite well be that certain schizophrenic patients do indeed develop this syndrome as a result of neuroleptic exposure to a brain which is dysfunctional in other ways. In other words, both the disease process and the neuroleptic exposure may be necessary, either one alone being insufficient. I have seen a number of cases in the long-term wards of state psychiatric centers who fit the description of the syndrome of tardive dysmentia.

• Finally, I find the authors’ speculation on “pseudo-manic depression” being mistakenly diagnosed as “true” manic depression particularly disconcerting. Nowhere in their study have they provided evidence to support this statement. The characteristic symptoms of mania such as hyperenergia, decreased need for sleep, pressured speech, flight of ideas, grandiosity, racing thoughts, or hyperactivity are not reported to be found in excess in patients with tardive dysmentia. In an American psychiatric practice setting, with a long tradition of favoring a diagnosis of schizophrenia over mania in psychotic patients, the authors are presenting an unsupported argument to caution against the rediagnosing of certain “schizophrenias” to bipolar disorder. This is even more unjustifiable in the light of their proposing a new neurotoxic iatrogenic syndrome as a result of treatment with drugs that are primarily used in the pharmacotherapy of schizophrenia.

While bold speculations open the way for new discoveries, these should be done with great caution and bearing in mind the evidence. The concept of tardive dysmentia—at present merely an interesting speculation—needs to be observed more carefully. Unlike the skeptics who denied the possibility of neuroleptic-related tardive dyskinesia in the early years following the first reports of its occurrence, I would urge careful and systematic inquiry into the nature of this syndrome and its putative relationship with neuroleptic exposure. We have just concluded a study of this proposed syndrome in 60 inpatients and will be reporting on our findings in the near future.

References

The Author
Sukdeb Mukherjee, M.D., is Clinical/Research Director of the Columbia-Creemoor Intensive Psychiatric Care Unit at Creedmoor Psychiatric Center, N.Y.; Assistant Attending Psychiatrist at New York State Psychiatric Institute, Department of Biological Psychiatry; and Assistant Clinical Professor of Psychiatry, at the College of Physicians and Surgeons of Columbia University.

The Author Replies:
Mukherjee (1984) has raised a number of questions concerning our recent article “Is There a Tardive Dysmentia?” (Wilson et al. 1983). I would like to respond to several of his points and make some general comments as well.

In regard to our methods of diagnosis and assessment of tardive dyskinesia (TD), we were guided by the literature at the time the study was completed in 1978. The more recent diagnostic criteria and means of interpreting rating scales Dr. Mukherjee refers to should further enhance research in TD.

Concerning the relatively high
The prevalence rate of TD in our study, it is true that our sample was biased in that it consisted of chronically ill and institutionalized patients who had been on neuroleptics an average of 17 years. We chose this population because it represented individuals with the highest risk of TD. We would also advocate caution in making any generalizations from our study.

The key question that Dr. Mukherjee raises is whether the reported phenomena are neuroleptic related, schizophrenia related, or related to some combination of the two. As evidence against a unitary neuroleptic-induced process, he describes data that he has gathered on 75 bipolar patients, some of whom had neuroleptic-induced TD, but none of whom showed "dysmentia" behaviors when well. He rightly points out that this observation neither supports nor rules out the hypothesis of a neuroleptic-induced "dysmentia." It does, however, argue against the hypothesis of a one-to-one association between TD and "dysmentia" behaviors. The literature on TD would also clearly seem to support this since no consistent nonmotor behavioral changes have been reported in patients with TD. Whether the phenomena are related to an interaction between neuroleptics and schizophrenia, to schizophrenia itself, or to another variable such as institutionalization remains to be determined. In fact, much work will be required simply to assess whether neuroleptics do in fact induce long-term behavioral changes other than TD. I am in full agreement with Dr. Mukherjee that these issues require careful study.

The issue of whether patients with tardive dysmentia could meet full criteria for manic-depressive illness or not was not meant to be a main point of the article. We were more interested in suggesting that the "dysmentia" process might lead to affective changes with an excited component, and that this in turn might lead clinicians to question the diagnosis of schizophrenia.

The late Dr. Wilson developed a concept of neuroleptic-induced behavioral changes in schizophrenia after many years of clinical observation. Our reported observations represented an initial presentation of his idea, and one that we hoped would stimulate thought. Clearly, the latter goal appears to have been achieved.

References

The Author
James C. Garbutt, M.D., is Assistant Professor of Psychiatry, Department of Psychiatry, University of North Carolina, School of Medicine, Chapel Hill, NC 27514.

An Invitation to Readers
Providing a forum for a lively exchange of ideas ranks high among the Schizophrenia Bulletin's objectives. In the section At Issue, readers are asked to comment on specific controversial subjects that merit wide discussion. But remarks need not be confined to the issues we have identified. At Issue is open to any schizophrenia-related topic that needs airing. It is a place for readers to discuss articles that appear in the Bulletin or elsewhere in the professional literature, to report informally on experiences in the clinic, laboratory, or community, and to share ideas—including those that might seem to be radical notions. We welcome all comments.—The Editors.

Send your remarks to:
At Issue
Center for Studies of Schizophrenia
National Institute of Mental Health
Alcohol, Drug Abuse, and Mental Health Administration
5600 Fishers Lane, Rm. 10C–16
Rockville, MD 20857